

per fibre-year of cumulative exposure for the two New Orleans plants; the values should be 0.031 and 0.100. This comparison considers only a single, combined measure of observed risk; in comparing only these three cohorts more detailed information is available and should be used. Moreover, this comparison relies on the exposure estimates for the Canadian cohort, which have been questioned, since no sensible pattern of lung cancer risk with cumulative exposure was observed for that cohort. In comparing these three cohorts, attention may be restricted to workers in our plants who were employed for more than five years. With median durations of more than 15 years for these two groups, they are reasonably comparable with the Canadian cohort, with a median duration estimated as approximately ten years. Despite similar average exposure concentration estimates, the lung cancer results remain surprisingly different: SMRs of 109 and 221 for these subgroups in our plants compared with 490 for the Canadian cohort. These differences, even those between the two New Orleans plants, suggest important differences in these plants, possibly in the actual exposure concentrations or in fibre types.

With regard to causes of death in our residual cancer category, we pointed out the number of deaths for each of the principal ICD codes within this category precisely because of our concern about this group. We do not agree, however, that the "best evidence" approach is appropriate here since we are primarily interested in comparisons with general population rates based on death certificate information. Internal comparisons can show a dose response relation, thus establishing causality, but this is hardly necessary for asbestos and lung cancer today.

We may, however, consider this group in another way. Since there are ICD codes for secondary digestive/respiratory cancers and for cancer with site unspecified, comparisons were made of the observed and expected numbers, as for any other sites. In neither plant were the secondary digestive/respiratory cancers raised compared with Louisiana rates. For site unspecified among plant 1 workers, there was a small excess among the shortest term workers but no excess in the longer term and no trend with cumulative exposure. In plant 2 there were 26 cancers with unspecified (including three mesotheliomas) compared with 14.2 expected. The possible concern, therefore, is with the 11.8 excess cases with site unspecified, and whether some of these could be lung cancers. In an attempt to determine how much lung cancer dose response would be affected if some of these excess site unspecified cancers were actually lung cancers for each cumulative category of asbestos exposure the excess cases were distributed to the specific sites (lung or digestive, for example) in the same proportion as the cases of cancer with site

specified. This allocation had only a minimal effect on the non-respiratory cancer SMRs. The weighted least squares regression line for lung cancer and cumulative asbestos exposure, without forcing an intercept of one, became  $1.24 + 0.0075 \times$ , for  $\times$  in f/ml-y (the slope was statistically significant,  $p < 0.02$ ). This compares with the reported fit of  $1.17 + 0.0061 \times$ , without allocation of these cases. Thus the cancers of unspecified sites had little effect on the dose response relation; the fit using a forced intercept of 1.0 was also not changed appreciably by allocating these cases.

Finally, we are amazed at Finkelstein's gratuitous remarks concerning "blue collar workers in the southern United States" and the "quality of medical care under the free enterprise American health care system," implying that misdiagnoses may be related. There is not a whit of evidence to support this socioeconomic speculation and we find such commentary inappropriate. Moreover, the State of Louisiana has long been a recognised leader in providing free medical care to its citizens; the primary facility of this system is located in New Orleans and serves as a teaching hospital for two university medical schools.

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## Asbestos related lung disease in maintenance workers

SIR,—Hilt's recent study (1987;44:621-6) of the prevalence of non-malignant asbestos related lung disorders in a cohort of chemical industry workers exposed to asbestos highlights two important aspects of asbestos related occupational lung disease: (1) the importance of "indirect" or "secondary" exposures in the aetiology of these diseases and (2) the need for increased recognition of such exposures and monitoring of these occupational groups.

Indirect exposure to asbestos in occupational settings is becoming an increasingly recognised health risk, particularly in non-asbestos industries. For instance, Paci *et al* have described recently the existence of an asbestos hazard among non-asbestos textile workers in Italy, specifically among "rag sorters" working in the reprocessed textile industry.<sup>1</sup> These individuals were exposed to asbestos fibres freed from polypropylene bags previously used to transport asbestos. These bags were cut and used in the textile

plants to cover bales of rags before shipment. A possible asbestos related cancer risk may also exist in other non-asbestos occupational setting previously thought to present little risk to workers.<sup>2,3</sup>

Hilt also raises the intriguing question of asbestos disease risk among "maintenance workers." Lilis and her co-authors studied chemical industry maintenance workers<sup>4</sup> and she has recently co-authored a study of maintenance personnel employed by the New York City Board of Education.<sup>5</sup> This study was conducted to evaluate the health status of those employees considered by the Board potentially to have had occupational exposure to asbestos materials. Of 115 workers examined, 23% had x ray abnormalities consistent with exposure to asbestos. A significant burden of asbestosis was found to exist among this group even when individuals with previous shipyard exposure were excluded (26%).

The United States Environmental Protection Agency estimates that about 30 000 school buildings across the United States contain friable asbestos. These materials may pose an important risk to the health of a large workforce of maintenance personnel previously thought not to be at risk. As suggested by Hilt's study, continued surveillance of such groups will provide the important information necessary to define clearly the risk of developing non-malignant and malignant asbestos diseases due to indirect asbestos exposure.

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## Investigating dose response relations on occupational mortality studies

SIR,—Swaen and Volovics in this journal (1987;44:642-4) and Whittemore in the *American Journal of Industrial Medicine*<sup>1</sup> have independently discussed an approach to the calculation of dose response relations in occupational mortality studies that looks at the problem of determining risk when employment and observation times overlap. These authors have pointed out that risk among workers with longer employment times may be underestimated, compared with workers with briefer employment, if all of the person-years of observation used to calculate SMRs are attributed to the longest employment category. To avoid an artefactual flattening of the dose response relation, they propose a method by which person-years of observation are left behind in exposure categories pertaining to shorter periods of employment as a worker continues in employment and subsequently moves into categories of longer employment. Distributing person-years in this manner is an improvement over the method that attributes person-years to the highest exposure category, but invalid inferences may still be drawn for those diseases such as lung cancer and mesothelioma in asbestos workers in which the risk of disease varies with the length of time from first exposure.

Implicit in the calculation of the SMR is the assumption that the risk among any subgroup of workers is a constant multiple of the underlying risk in the comparison population. If this assumption does not hold the SMR is uninformative as a summary measure. Whittemore cites an extreme example in which an occupational exposure increases the death rate among men but decreases it among women. An overall SMR for both sexes combined is uninformative here and sex specific SMRs are required. In the case of many occupational exposures the annual risk of disease is not constant, but varies with the period from first exposure. Asbestos associated lung cancer, for example, does not usually become apparent until 15 or more years from first exposure whereas the incidence of mesothelioma increases with the third or fourth power of time from first exposure. In situations such as these a worker's risk, relative to that in the reference population, varies with time and the multiplicative assumption underlying the SMR is violated unless latency specific comparisons are made.

What is the effect of applying the proposed method of leaving behind person-years as workers move into longer employment categories? By logical necessity, the person-years left behind will be of shorter "latency" than the years contributed to longer employment categories, and the "average latency" will tend to increase from one exposure grouping to the next. If